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on

INVESTIGATIONS ON THE ANTIGENICITY OF SNAKE VENOMS

submitted by

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In the Annual Report for the First Year of Study we have reported on our attempts to produce a potent antivenin against *V. palaestinae* venom in horses. At that time our attempts were not very successful and the horse antiserum was of very poor antibody titer. Continued immunization of horses with whole *Vipera palaestinae* venom, using large amounts of venom, were found ineffective for up to a period of 17 months. The neutralizing capacity of 1 ml serum did not exceed 10 LD₅₀ of whole venom; this was mainly against the hemorrhagic fraction and no neutralization of the neurotoxic fraction whatsoever was obtained.

At that time, a major change was introduced in the immunization procedure through the use of venom-adjuvant mixtures. A single injection of a venom-adjuvant mixture (containing 100 mgm of venom) resulted in a considerable rise in the neutralizing capacity of the serum against whole venom (110 LD₅₀/ml) and "hemorrhagin" (130 LD₅₀/ml). This enhancement of the immunogenicity of *Vipera palaestinae* venom by using a venom-adjuvant mixture was similar to that found previously for the hemorrhagic *Echis colorata* venom (see: Quarterly Technical Status Report No. 1).

In contrast to the marked rise in anti-whole venom and anti-hemorrhagin titers, the venom-adjuvant mixture, although containing a considerable amount of neurotoxin, induced little or no antineurotoxic antibodies. Evidently, as pointed out previously, the neurotoxin molecule is of low immunogenic potency, presumably due to its low molecular weight (mw = 12,000). However, when 10 mgm of carboxy-methyl-cellulose (CMC) - bound neurotoxin were administered, a marked rise in antineurotoxin titer was obtained, reaching a value of neutralizing capacity of

50 LD₅₀/ ml serum 10 days after the injection. It is noteworthy, that whereas this rise in antineurotoxic potency of the serum was reflected in a parallel rise of neutralizing capacity against whole venom, no increase in antihemorrhagic titer was observed. This result obtained in horses illustrates well the previously reported enhancing effect of binding the neurotoxin to CMC on its immunogenicity, first demonstrated by us in rabbits. It is intended to follow the antivenin titers, both antihemorrhagic and antineurotoxic, during the next few months and accordingly administer booster injections.